

Asbestos-related Radiographic Abnormalities in Elevator Construction Workers

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Elevator construction workers are exposed to asbestos dust during construction and refurbishment work on older buildings. We screened a cohort of workers, all with greater than 20 yr of employment in the industry, with clinical examinations, chest radiography ("B" reader interpretations), and routine spirometry. Twenty of the 91 workers (22%) had evidence of pleural disease, but none of them had an interstitial process consistent with asbestosis. Of those with pleural thickening, 15 had bilateral circumscribed plaques and five had unilateral plaque formation. There were no cases of diffuse pleural thickening, benign pleural effusions, or mesothelioma identified in our cohort. The difference in the mean body mass index of those with pleural abnormalities (29.18 ± 3.95) and those without (27.7 ± 3.86) was not statistically significant ($p = 0.135$). We conclude that elevator construction workers have an increased risk for the development of asbestos-related pleural disease.

Recent reports have identified various nonmanufacturing occupational groups whose members are at risk for contracting asbestos-related disease. These groups include shipyard workers (1), boiler-makers (2), electricians (3), railroad workers (4), and sheet metal workers (5, 6). One group of workers that has not as yet been identified in the literature as being at risk for asbestos-related disease is elevator construction workers.

Prior to 1973, asbestos was sprayed in parts of buildings for insulation purposes. In 1973, when the U.S. Environmental Protection Agency banned the spraying of asbestos for insulation in construction, more than half of the high-rise buildings in the United States contained sprayed asbestos insulation (7, 8). Elevator workers, especially those who work in the areas of construction, modernization, and service, are at risk for asbestos exposure. This risk for exposure comes from asbestos-sprayed girders and other structures in elevator shafts of these pre-1973 constructions. This report summarizes the chest radiographic abnormalities in a previously screened cohort of elevator workers.

METHODS

Population

One of the authors (EB) conducted a cross-sectional screening of the Philadelphia Local of the International Union of Elevator Constructors (IUEC). Workers who had been members of the union for at least 20 yr were eligible for inclusion in the survey. Participation was voluntary and open to currently employed, unemployed, and retired members. The union

decided to keep the total number of eligible workers and their demographic data confidential, and they would not release this information. Ninety-one (eight retired) men agreed to participate in the screening in November 1988, which included a questionnaire on work and health data, spirometry, and a chest radiograph. As this report is a retrospective confidential analysis of previously collected data, consent for chart review was not obtained from screening participants. The Committee for the Protection of Human Subjects does not require informed consent for this type of study.

Pulmonary Function Tests

Maximal expiratory volume-time curves were obtained in the sitting position using an Eagle II survey spirometer (Warren E. Collins, Braintree, MA). A technician certified by the National Institute for Occupational Safety and Health obtained at least three curves from each participant. Seventy-eight of 91 patients (86%) had reproducible and acceptable curves according to ATS standards (9). The remaining patients all had at least one acceptable curve, and they were not excluded on the basis of nonreproducibility of tracings. The highest values for FVC and FEV₁, after correction for BTPS, were used in the analysis. The system was calibrated at the beginning of each half day of the screening using a 3.0-L calibration syringe.

Predicted values based on age, sex, race, and height were calculated using equations derived by Crapo and coworkers (10, 11).

Questionnaires

All participants in the screening completed a questionnaire assessing their work as elevator constructors. Information was obtained on the number of years worked in the trade, the degree of asbestos exposure, and the last and the first years worked as an elevator constructor. Duration of exposure was calculated as the difference between the latter two values. In addition, participants completed a modified Epidemiology Standardization Project questionnaire to assess respiratory symptoms and smoking history.

Chest Radiograph

Posterior-anterior chest radiographs were obtained at maximal inspiration. Each film was first interpreted independently by two "B" readers (EB, MG) using the International Labour Office (ILO) 1980 classification system (12). A third "B" reader (WG) evaluated 43 films that were considered to possi-

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TABLE 1

DESCRIPTIVE STATISTICS FOR 91 SCREENING PARTICIPANTS*

	100
Age, yr	68.9
Years as elevator constructor	52.2 ± 7.9
Smoking history	27.1 ± 5.8
Smoking history	16.5
Smoking history	47.2
Smoking history	36.3

All values are expressed as the mean ± standard deviation or as a percentage of all cases.

have any abnormality consistent with a pneumoconiosis. When only one of the first two readers found evidence of any plaques or interstitial changes greater than 1/0, agreement by two of the three readers constituted the final reading. All three readers interpreted each film without knowledge of the exposure, the medical history of the worker being examined, or the reading of the other "B" readers. Films were examined in random order without the inclusion of nonexposed control films.

Chest radiographs were classified for parenchymal abnormalities using the ILO system. The films were also classified as showing pleural thickening if either unilateral or bilateral pleural thickening was present and consistent with the diagnosis of pneumoconiosis.

Statistical Analysis

Group comparisons between numeric variables were performed using two-tailed t tests (for two groups) or ANOVAs (for more than two groups). A t test between two groups with 45 subjects in each would have 80% power to detect a difference of 0.6 standard deviations between the groups.

When two groups needed to be compared on an ordinal variable (e.g., smoking status: never, former, current), this was most often done by treating the variable as the outcome and computing a single degree of freedom chi-square test of trend (slope) across the levels of the ordinal variable.

RESULTS

Descriptive data about the participants are listed in table 1. None of the chest radiographs of the study participants were classified as having greater than or equal to 1/0 interstitial opacities. A total of 20 of the 91 participants (22.0%; 95% confidence interval: 14–33%) showed evidence of pleural abnormalities, of whom fifteen had bilateral pleural thickening, whereas five had unilateral pleural thickening. No participant was classified according to ILO criteria as having diffuse pleural thickening.

All three "B" readers agreed on 12 of the 15 classified as having bilateral plaques. In one case where the plaque was classified as bilateral, the third reader thought the plaque was unilateral.

In the remaining two cases, one of the initial readers thought there were no plaques.

Of the five participants classified as having unilateral plaques, the three readers agreed in only one case. In three of the participants, the initial readers agreed on unilateral plaques, but the third reader thought there were bilateral plaques (two cases) or no plaques (one case). In the fifth participant, one of the initial readers thought there was no plaque.

All but three of the radiographs with pleural thickening were classified as chest wall width A, extent 1 or 2. Five showed calcifications of the chest wall or diaphragm.

We assessed the association between years worked as an elevator constructor and the presence of pleural abnormalities. The difference between the sample means of total years worked between those with any pleural thickening (29.2 yr) and those without any pleural thickening (26.5 yr) was small, and it was not statistically significant when considered alone ($p = 0.067$ level) nor when controlling for age ($p = 0.465$). Moreover, the difference in sample means of years worked as an elevator constructor between those with and without bilateral pleural thickening was not statistically significant ($p = 0.122$). There was also no difference in the prevalence of pleural thickening when the cohort was divided into three strata based on years of employment as an elevator constructor.

There was no association between pleural abnormalities and cigarette smoking. However, there was a statistically significant association between the prevalence of chronic bronchitis, obstructive impairment, and smoking (data not shown).

There were no significant differences in mean spirometric values between those with and without either bilateral pleural thickening or any pleural thickening (table 2). With unequal sample sizes (20 versus 71), there would have to be approximately standard deviations of difference for 80% power. This is an absolute difference of about 12 in mean percent predicted values for FEV₁ and FVC. The difference in the mean body mass index (BMI) of those with pleural abnormalities (29.18 ± 3.95) and those with normal chest radiographs (27.7 ± 3.86) was not statistically significant ($p = 0.135$).

DISCUSSION

The adverse health effects of chronic asbestos exposure in high risk occupations such as insulators and shipyard work has been extensively reviewed in the scientific literature (13, 14). However, several cohorts in the construction industry such as elevator construction workers have not been evaluated previously for the poten-

TABLE 2
RELATIONSHIP BETWEEN PLEURAL ABNORMALITIES AND PULMONARY FUNCTION*

	FEV ₁ (L)	FVC (L)	FEF ₂₅₋₇₅ (L/s)	FEV ₁ /FVC
Pleural thickening				
Bilateral, n = 15				
Unadjusted	3.33 ± 0.63	4.15 ± 0.72	3.11 ± 1.35	
% Predicted†	86.5 ± 13.4	85.0 ± 11.7	82.6 ± 40.5	80.2 ± 6.9
Bilateral and unilateral, n = 20				
Unadjusted	3.33 ± 0.56	4.20 ± 0.66	3.30 ± 1.33	
% Predicted†	86.3 ± 11.8	85.8 ± 10.6	80.7 ± 40.0	79.5 ± 7.7
No pleural abnormalities, n = 71				
Unadjusted	3.40 ± 0.82	4.43 ± 0.81	3.07 ± 1.31	
% Predicted†	86.1 ± 19.7	88.4 ± 16.2	79.5 ± 32.1	76.4 ± 11.6

* All values are mean ± SD.

† Predicted values based on prediction equations derived by Crapo and coworkers (10).

consequences of their lower intensity, secondary occupational asbestos exposure.

Twenty-two percent (20 of 91) of our screened participants had radiographic evidence of pleural abnormalities that were considered by "B" criteria as consistent with pneumoconiosis. There were 10 participants with bilateral pleural plaques/thickening and five with unilateral plaque formation. There were no examples of diffuse pleural thickening (as defined by ILO criteria), mesothelioma, benign pleural effusions in our cohort. The generalizability of these results is limited by the fact that our cohort did not include active, retired, former, or deceased elevator workers. The direction of bias is difficult to predict, however. Active, nonparticipating workers may be healthier than screening participants, whereas retired, former, or deceased workers may be more diseased as a group. As a result, our analysis may estimate inaccurately the true prevalence of disease.

Although we did not screen an unexposed control group, the baseline prevalence of pleural plaques/thickening has been estimated to vary between 0.21% in a blue collar population (15) to 5% in a university employee population (16, 17). Therefore, we considered the prevalence of pleural disease in our cohort to be initially significant and, interestingly, comparable in frequency to reported screenings of pipefitters and plumbers (18), sheet-metal workers (5), electricians (3), and public school custodians (19).

We excluded from our analysis any patients in whom the pleural changes were considered to be caused by rib fracture, postoperative or radiation effects, trauma, tuberculosis, or empyema. Because chest CT scans were not obtained, it could be argued that the pleural thickening was related to obesity and fat deposition along the lateral chest wall (17). In an attempt to assess this possibility, we compared BMI in the pleural disease group with that in the nonpleural disease group. If obesity were a factor in the prevalence of pleural changes, it would have been expected that the BMI would be higher in the pleural disease group. However, our results demonstrated no significant difference between these groups. Consequently, the pleural changes in our cohort were considered, by exclusion, as asbestos-related phenomenon secondary to chronic occupational exposure.

In contrast to other investigators (18), we did not find a significant relationship between duration of exposure and the prevalence of pleural disease in building construction workers. This finding was likely due to a selection bias as we screened only elevator constructors with 20 yr or more of union membership. By eliminating those workers with fewer years of exposure from our analysis, we were unable to demonstrate a direct association between duration of exposure to asbestos and prevalence of pleural thickening noted in other screening programs.

To determine if the pleural abnormalities produced any impairment in pulmonary function, we compared the spirometric indices of FEV₁, FVC, FEF₂₅₋₇₅, and FEV₁/FVC between those with and those without pleural disease (table 2). Unlike Schwartz and coworkers (6, 20) and others (21-26) who reported an association between asbestos-related pleural plaque and decrements in lung function, our findings indicate that the pleural changes in our cohort of elevator construction workers were not associated with any significant mean functional changes compared with those in the no pleural disease group. It should be noted, however, that the percent predicted FVC in other investigations of asbestos-related pleural plaque disease (21, 24) is similar to the percent predicted FVC in our pleural plaque group. Therefore, it is doubtful that the extent of the pleural changes were more extensive in other study groups than in our elevator construction workers. Rather, the difference in results may reflect either the relatively small number of

subjects in our screening and the low power when group means are not grossly disparate or the unexpectedly low normal FVC percent predicted in our no pleural disease group.

In summary, a screening of 91 elevator construction workers revealed that circumscribed pleural plaque formation was present in 22%. It is likely that indirect exposure to asbestos dust produced these radiographic abnormalities. We conclude that elevator construction workers, with 20 yr or more of union membership, have had a significant occupational exposure to asbestos as manifested by an increased prevalence of pleural disease. We recommend that the possible risks associated with this exposure should be acknowledged in the health screening and subsequent medical follow-up of this group of workers. At the very least, union leadership and relevant health professionals should encourage smoking cessation in this cohort to reduce the increased prevalence of smoking and chronic obstructive lung disease, and to reduce the potential synergistic effects between cigarettes and asbestos. In addition, employers and unions should take steps to reduce asbestos exposure of employees working in elevator shafts.

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